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Management of Severe Perioperative Bleeding

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Abstract

Perioperative bleeding continues to be a serious problem during and after surgery, leading to increased mortality and morbidity. Non-vascular sources of haemostatic perioperative bleeding often result from a bleeding problem that has gone undiscovered, from the specifics of the surgery itself, or acquired coagulation abnormalities due to haemorrhage, hemodilution, or the administration of haemostatic factors. Allogeneic blood product delivery, pharmacologic medicines, and the growing use of pure or recombinant haemostatic factors are all considered conventional treatment techniques in the management of bleeding patients. Trauma and sophisticated surgical operations, such as heart surgery and liver transplantation, cause a cascade of haemostatic alterations in the perioperative period. Tranexamic acid, desmopressin, fibrinogen, and prothrombin complex concentrates are some of the newer methods used for both the prevention and treatment of perioperative bleeding. More in-depth evaluation of targeted treatment for haemostasis is now possible because to point-of-care testing using thromboelastography, rotational thromboelastometry, and platelet function tests. Better management, less use of allogeneic blood products, and fewer transfusion-related complications may be achieved by multimodal, and strategic approaches. The goal of this effort is to alleviate the difficulty of controlling bleeding in surgical patients. Research priorities include, but are not limited to, preventing hypotension, sustaining appropriate tissue perfusion and oxygenation, and preventing bleeding.

Keywords: - Extreme Blood Loss During Surgery; Low Blood Pressure and Hemostasis.

1. Introduction

Bleeding in surgical patients has several causes including blood loss, haemodilution, acquired platelet dysfunction, coagulation factor consumption in extracorporeal circuits, activation of fibrinolytic, fibrinogenolytic, inflammatory pathways, and hypothermia [1].

Oral anticoagulants [warfarin, dabigatran, rivaroxaban, apixaban, edoxaban] and platelet inhibitors are a common cause of acquired haemostatic abnormalities in surgical patients [P2Y12 receptor inhibitors-clopidogrel, prasugrel, or ticagrelor]. Therefore, haemostasis abnormalities, either congenital or acquired, may contribute to postoperative bleeding. Patients with congenital bleeding problems are less prevalent, and if they appear for surgery, they have likely previously been treated. The International Society on Thrombosis and Haemostasis ISTH bleeding questionnaire is just as good as a battery of laboratory tests for predicting the likelihood of postoperative bleeding, and it may be used as part of a preoperative examination [2].

There is often just bleeding at the surgical site and is localised to that area. Limiting surgical bleeding in patients of high risk requires careful surgical skill, patience, and careful patient selection. A comprehensive analysis of the wide variety of topical haemostatic drugs and devices now on the market is beyond the scope of the present article [3].

Blood physiology

The life-sustaining component of blood must be present to keep a person alive. The circulating blood is essential because it carries oxygen, hormones, gases, and waste products throughout the body. Significant immunological roles are also played by it. The homeostatic control of pH, temperature and other interior factors all rely heavily on blood. In addition to plasma and erythrocytes, blood also contains platelets and leukocytes [white blood cells] [4].

Between 4.5 and 5 liters of cells and plasma-filled blood flow through the adult human circulatory system. About 55% of the volume of blood is plasma. The remaining 45 percent is made up of several cellular types. In an average, healthy adult, the blood volume accounts for just around 7–8% of the person's entire mass [5].

Haemorrhage

A haemorrhage occurs when a blood artery is broken and blood suddenly leaks out. It's possible for the bleeding to be little, like in the case of petechiae and ecchymosis caused by injury to the skin's superficial blood vessels. Changes in vital signs and changed mental state are only two examples of the more nebulous constellation of symptoms that might result [6].

Both internal and external bleeding is possible. When blood leaks from the body, it does so either via an orifice or a wound.

Clinical suspicion of internal bleeding involves a comprehensive history and physical examination, laboratory investigations, imaging, and vigilant monitoring of vital signs. Particularly among the acute trauma population, haemorrhage is a major contributor to avoidable mortality [7].

Preoperative bleeding may be caused by several factors, including: -

Trauma

Haemorrhage is the primary cause of mortality in patients who survive to hospital for admission after significant trauma, with the largest frequency occurring in the first three to four hours after arrival. Major trauma may be caused by a wide variety of events, such as car accidents, gunfire, falls from great heights, shards of glass, explosions, etc. Extreme blood loss may occur as a result of a ruptured organ [spleen damage] or significant vascular laceration [bleeding from the brain]. Massive haemorrhaging causes the majority of patients to die before they reach the hospital. Consequently, haemorrhage/hemorrhagic shock continues to be the primary cause of mortality in all significant traumatic injuries [8].

Potential Dangers of Surgery

There is a close relationship between the operational process and the ensuing danger. All studies that have looked at the topic agree that emergency surgery has a higher risk than planned procedures. The underlying illness processes and the stress of the surgical treatment might contribute to the risk of surgery in certain situations. In terms of mortality and serious complications, cardiovascular surgery has consistently ranked at the very top of the list of all types of major operations. Vascular surgery, one of the most dangerous types of surgery outside the heart, reviews the risks associated with anaesthesia for cardiac surgical operations. The burden of coronary artery disease may be larger in this group; however, studies have indicated that infra-inguinal treatments have a comparable incidence of cardiac morbidity as aortic reconstructive surgery [9].

Amputation is another example of a high-risk vascular operation. There is also a higher chance of complications after abdominal, thoracic, or orthopaedic surgery [10].

Unusual clotting and bleeding

Shock-induced coagulopathy: Several causes, including consumption of clotting factors, hemodilution from massive quantities of crystalloid infusion, acidosis, and hypothermia, are considered to have a role in this illness, which may be iatrogenic or secondary in nature. It has yet to be determined what causes coagulopathy. According to one view, thrombin and fibrin are created and used because of tissue factors released in response to damage [11].

It has also been theorised that trauma-induced hypoperfusion and ischemia trigger the production of activated protein C, which in turn causes the consumption of plasminogen activator inhibitor, blockage of the clotting cascade, systemic anticoagulation, and hyper-fibrinolysis. Acute traumatic coagulopathy is now treated with a high ratio of fresh frozen plasma to red blood cells [12].

Health issues related to pregnancy and childbirth

Haemorrhage during pregnancy is a leading cause of death for mothers. Large-scale bleeding in pregnant women may happen before, during, or after giving birth. Intense bleeding during childbirth seems to be the leading cause of maternal death and disability across the globe [13].

Alternating physiological processes and the body's reaction to sudden blood loss.

One of the most crucial homeostatic processes is the maintenance of blood volume in circulation. Tissues in hemorrhagic shock do not get enough blood due to a drop in blood volume. So, when tissue perfusion is inadequate, organ failure rapidly develops, affecting every cell in the body. [14].

Stabilization and cellular regulation at the subcellular level

Extreme blood loss triggers a cascade of stress reactions that direct blood flows to essential organs and tells cells to burn up stored energy. The baroreceptors are triggered by the decreased wall tension in the major intrathoracic arteries caused by hemorrhagic shock. Adrenergic responses are triggered, which include both neuronal and circulatory hormonal components. The effects on the nervous system are almost instantaneous, whereas the hormonal alterations may occur quickly but take some time to manifest. Stellate ganglion sympathetic fibers stimulate the heart, while regional ganglia sympathetic fibers generate peripheral artery vasoconstriction; these are the two main neurological components [15].

Endocrine Reaction: Stress hormones in the bloodstream are produced primarily by the hypothalamic-pituitary-adrenomedullary axis. They cause the adrenal glands to release

hormones such as adrenaline and norepinephrine, corticosteroids from the adrenal cortex, renin from the kidneys, and glucagon from the pancreas. These hormones induce the breakdown of tissue glycogen reserves, signal the liver to break down glycogen to release glucose into the plasma, and enhance the release of fatty acids from adipose tissue through lipolysis [16].

Lactate generation from skeletal muscles due to aerobic glycolysis may also occur in the resuscitated haemorrhagic shock patient. Therefore, it is clear that high lactate levels in the blood are a sentinel indication of systemic hypoperfusion. When proper resuscitation is performed, lactate levels normalise [17].

The body's reaction to bleeding may be affected by factors such as the patient's age, general health, body temperature [hypo- or hyperthermia], and the medications they're taking. You must keep this in mind while using this label. Children and newborns do not fall into this category because they rely more on heart rate than on maintaining blood pressure to make up for blood loss. A fatal outcome is certain when a child's blood pressure drops [18].

Alternative Methods for Identifying Hemorrhagic Shock

According to the ATLS categorization, not all cases of hemorrhagic shock are equally apparent. When bleeding occurs, a change in arterial acid-base balance generally precedes a drop in cardiac output [CO]. Even, if pH and blood pressure are unaffected, the arterial and venous blood bicarbonate level and base deficit will drop early in bleeding [19].

Hemorrhagic shock may be differentiated from simple bleeding by looking for signs of widespread tissue hypoperfusion when the base deficit drops below a certain critical value. Suspect hemorrhagic shock in patients with tachycardia, a low base deficit, and poor urine output [20].

Molecular and cellular reactions to sudden blood loss

When the systemic oxygen delivery [DO2] falls below DO2crit, tissues switch to anaerobic sources of energy [also known as "compensated shock"]. As long as enough adenosine triphosphate [ATP] is produced from aerobic and anaerobic sources, the cell may continue to operate under these circumstances. When it comes to hypoxia, certain tissues are more robust than others. Irreversible damage to isolated hepatocytes does not begin until 2.5 hours of ischemia, but skeletal and smooth muscles are very resistant to hypoxia. However, even brief exposure to oxygen deprivation is enough to irreparably harm brain cells [21].

It would suggest that the stomach is especially vulnerable to hypoperfusion. Before declines in systemic volume O2 [VO2] are identified, the intestine and stomach mucosa exhibit indications of anaerobic metabolism. When the body's aerobic and anaerobic ATP sources aren't enough to keep cells functioning, shock sets in and the damage to tissues is permanent. When ion transport pumps connected with the membrane fail, the cell membrane degrades and the cell swells. These pumps are especially important in the control of calcium and sodium [22].

Energy depletion, acidosis, free radical production, and adenine nucleotide loss are all factors that contribute to permanent cellular damage in hypoxia [23].

Controlling severe blood loss during surgery What Is Fluid Therapy?

Reducing blood loss, replenishing depleted blood volume, and restoring tissue perfusion and organ function are all objectives of fluid resuscitation. Systolic blood pressure [SBP] goals of 60–70 mmHg may be recommended for penetrating trauma; 80–90 mmHg for blunt trauma without traumatic brain injury [TBI]; and 100–110 mmHg for blunt trauma with TBI. Adhering to evidence-based clinical practice standards and modifying according to local therapy and the patient’s condition is likely to enhance patient outcomes despite the complexity and variability of clinical circumstances [24].

Only if the administered fluids lead to an increase in stroke volume [SV] and, in turn, cardiac output are they helpful. If the patient's SV rises by at least 10% after receiving a crystalloid fluid challenge of 500 mL, the patient is termed fluid responsive. Some dependable indicators of fluid responsiveness are pulse pressure fluctuation, the passive leg-raising test, and SV variation [25].

Examples of Infused Fluids Crystalloids, colloids, and blood are the three types of fluids. Each kind of fluid has its own set of benefits and uses in medical treatment. Crystalloid and colloid treatment are the main topics of this article [26].

Crystalloids are electrolyte solutions that may be used to replenish fluids lost via breathing and urination. Although crystalloids may temporarily enhance hemodynamics by increasing vascular volume, they are not as effective as colloid solutions. The chemical makeup and osmolality of crystalloids are two distinguishing characteristics. When compared

to plasma osmolality, both normal saline [NS] and lactated Ringer's [LR] are somewhat hypertonic [308 mOsm/l] and hypotonic [273 mOsm/l], respectively. With an osmolality of just 294 mOsm, plasmalyte is the most neutral electrolyte solution available [27].

Colloid solutions, which are more likely to stay in the intravascular compartment, have been proposed to treat hemorrhagic shock. Human albumin, hydroxyethyl starch [HES], and dextran are only a few examples of colloid solutions that have been tested in therapeutic settings. It takes less volume of resuscitative fluid to achieve hemodynamic stability when using a colloid solution than when using a crystalloid solution because colloid solutions are rapidly resuscitated after entering the intravascular compartment. However, the extracellular fluid volume deficit may be further compromised rather than restored by colloid solutions, which are more costly, may bind and reduce serum ionised calcium, and may reduce circulating levels of immunoglobulins. Crystalloid and colloid fluid resuscitation have both been the subject of much clinical and experimental research [28].

There is evidence from both clinical and experimental investigations to suggest that a modest amount of hypertonic saline [5 ml/kg NaCl 7.5%] with or without dextran may be an effective first resuscitation solution. Despite their hypertonicity, hypertonic solutions have no negative effects on immunological processes and have been shown to increase microvascular flow, reduce intracranial pressure, and stabilise arterial pressure and cardiac output with a minimal infusion volume [29].

It was determined that a 250 ml bolus of 7.5% saline delivered by a rapid infusion system should be used for the initial fluid resuscitation of the haemorrhaging battlefield casualty due to the safety and efficacy of hypertonic saline, the need for simplicity, the limited volume that can be carried in the field, especially in military scenarios, and the relatively low cost. An intraosseous needle or an intravenous catheter might be used to get systemic access. However, new clinical trial research has questioned this approach [30].

Pharmaceuticals for Resuscitation

To enhance the power of myocardial contraction, inotropes are a class of drugs that modify the heart's contractility. An inotrope may be an adrenergic agonist, phosphodiesterase III inhibitor, or calcium sensitizer [31].

Positive inotropic effects are produced by adrenergic agonists by activating beta-adrenergic receptors. They boost the heart rate, stroke volume, and carbon monoxide. Based on how they affect blood pressure and heart rate, adrenergic agonists [dobutamine, dopamine, norepinephrine, and epinephrine] may be broken down into subgroups [inopressors or inodilators]. In the United States, the only two inodilators on the market are milrinone and dobutamine [32].

Dopamine acts on the cardiovascular system via four receptors: dopaminergic type 1 and 2, and adrenergic alpha 1 and beta 1 receptors. Vasodilation of coronary, renal, and splanchnic arteries occurs at lower dosages [2.5 g/kg/minute]. Significant inotropic and chronotropic effects are mediated by beta-1 receptors of cardiomyocytes at dosages between 3 and 5 g/kg/min. It strongly constricts blood arteries through alpha-1 adrenergic receptors at high dosages [>5 g/kg/minute]. Extreme hypertension and tachyarrhythmia may result from such large dosages. Heart rate [HR], blood pressure [BP], and carbon monoxide [CO] are all boosted by dopamine and epinephrine due to their powerful beta-adrenergic action. The optimal range for dopamine's effects is between 5 and 10 g/kg/minute, while larger dosages cause a greater degree of vasoconstriction. Dopamine has a half-life of less than 2 minutes; thus, dosage modifications are usually not essential in cases of renal failure [33].

Phosphodiesterase III is an enzyme that milrinone blocks. Cyclic adenosine monophosphate is broken down by the phosphodiesterase III enzyme. If this enzyme is inhibited, cyclic adenosine monophosphate levels rise, leading to an increase in calcium influx channel phosphorylation. This causes a rise in intracellular calcium, which in turn stimulates actin-myosin cross-bridging and, ultimately, greater myocardial contractility. Through its effect on the vascular bed, it also promotes peripheral vasodilation by blocking the activation of myosin light chain in the smooth muscles of the blood vessels. In patients with heart failure or renal impairment, the half-life of milrinone is 2.3 to 2.4 hours. If your creatinine clearance is between 10 and 50 mL/minute, start milrinone at a dosage of less than 0.0625 to 0.125 mcg/kg/minute. Hypotension may occur with milrinone dosages >0.5 g/kg/minute [34].

In the cardiomyocyte, levosimendan acts as a calcium sensitizer, boosting cardiac contractility by making troponin C more sensitive to intracellular calcium. Because neither milrinone nor levosimendan use the beta-adrenergic route in their actions, they are favoured over beta-adrenergic inotropes in patients taking beta-blockers. In addition to

having a positive inotropic effect, levosimendan also produces peripheral vasodilation by activating ATP-sensitive potassium channels on the smooth muscle cells lining the vasculature. The active metabolite of levosimendan has a half-life of 70–80 hours. Its half-life is lengthened by renal dysfunction. In clinical practice, the medication's extended half-life is exploited by administering the drug in periodic pulse doses, since its effects are observed to remain even after 24 hours have passed after dosing was discontinued. The US market does not carry it. Since the renal clearance and half-life of milrinone and levosimendan are both prolonged, their steady-state effects are also delayed [34].

In order to enhance systemic vascular resistance and the MAP, vasopressors cause peripheral vasoconstriction by increasing intracellular calcium in the vascular myocyte. Catecholamines are widely regarded as a crucial component of therapy for cardiogenic shock. Up to 90% of those with cardiogenic shock need the use of vasopressor drugs. In the treatment of cardiogenic shock, they are recommended by the European Society of Cardiology as a class IIb/c and class IIb/c option, respectively [35].

Norepinephrine, epinephrine, dopamine, and phenylephrine are all examples of catecholamine vasopressors that are widely utilised. Vasopressors raise systolic and diastolic blood pressure by increasing the resistance of the blood vessels and the calcium in the cytoplasm of the myocytes that line the blood vessels. All of these things elevate mean arterial pressure MAP by activating adrenergic 1 receptor. Because of the challenges involved, there is a lack of clinical data from randomised studies comparing inotropes with vasopressors. The meta-analysis, expert opinion, and review papers are now used to suggest when and how to utilise vasopressors [36].

Precautions should be taken during surgery and anaesthesia for the treatment of severe bleeding

Initial laparotomy with the temporary closure of the abdomen, secondary resuscitation, and planned reoperation with final organ repair make up the damage control sequence in surgery. A methodical, collaborative strategy is required to finish the process successfully [37].

Surgeons should move quickly to finish the first laparotomy after deciding to use the damage control method. Hematoma and intraperitoneal pollution evacuation come first, followed by any required organ repairs. Clamps, sutures, or shunts may be used to manage major vascular injuries. During exploration and revascularization, it may be required to temporarily occlude the aorta to reduce bleeding. Suturing or stapling damage to hollow organs provides temporary relief. Stapling the ends of the bowel together creates a discontinuity when intestinal resections are done. When there is continuous bleeding, laparotomy pads may be put on top. To reduce intra-abdominal pressure and protect fascial integrity, the abdominal skin is temporarily closed by reapproximating the skin margins using towel clips or sutures [38].

There may be no way to close the abdomen if fluid resuscitation has caused significant visceral edema. Vacuum-assisted devices, X-ray cassette covers, sterile intravenous bags, and absorbable mesh are all excellent choices in this situation [39].

Restoration of normal physiology and treatment of the underlying coagulopathy constitute the second phase of damage control. Once the surgery is complete, the patient is sent to the ICU for further life-saving measures. Radiant heat lighting, heating blankets, and chest tube insertion with warm saline pleural lavage may all be used to rewarm the patient's core. A quick infusion device may be used to give patients warm blood, crystalloid, or plasma. Electrolyte, haematology, and coagulation profiles should be monitored on a regular basis in the laboratory and aggressively corrected as needed. Adequate mechanical ventilation and constant monitoring of hemodynamics are essential [40].

After the patient has been rewarmed, the coagulopathy has been corrected, and the fluid status and hemodynamics have been optimised, the patient is brought back into the operating theatre to have the packs removed and the final procedure performed. This is often done between 24 and 72 hours following the first procedure. Peritoneal contents should be reexamined for injuries or nonviable intestine segments once all laparotomy pads have been removed. Regular anastomotic methods, including the formation of an endostomy if necessary, may be used to restore intestinal continuity. Insertion of an enteral feeding tube might be considered if mechanical breathing is expected to be required for an extended period of time. After the procedure is done, the abdominal fascia is closed in the usual manner. Absorbable mesh or a vacuum-assisted closure device should be used if the fascia cannot be approximated tension-free [41].

Damage control surgery has a high risk of serious complications and even death, thus it's crucial that only the healthiest people do it.

Wound infection, intra-abdominal abscess, wound dehiscence, bile leak, entero-cutaneous fistula, multiple-system organ failure, and abdominal compartment syndrome are all common consequences [39].

When administering anaesthesia, there are several special concerns for trauma patients.

Pre-induction

The deadly trifecta of coagulopathy, acidosis, and hypothermia include low body temperature as one of its components.

Therefore, it is crucial to have a warmed intravenous [IV] line, a forced air warmer, and a fast infuser with warming capabilities on hand, and to warm the operating room to higher than 30°C.

Induction of anaesthesia

Standard inspections ensure that life-saving equipment is available for rapid use [e.g., anaesthetic machine check, verification of airway equipment, drugs, and special instruments] [42].

The patient is exsanguinating, and inducing anaesthesia might be fatal. Continual resuscitation of volume to forestall this from happening is essential. The time between identifying a patient for surgery and establishing vascular access [intravenous or intraosseous] and placing monitoring equipment [oxygen saturation, blood pressure, and ECG] is crucial. A patient in extremis should not have to wait to be induced so that, central access may be placed or invasive monitoring can begin. The time needed to place monitors during the surgical prep and drape may be reduced by doing so simultaneously. Having the patient lie on their back with their arms out allows for maximum surgical exposure and easy access to the arms when required throughout the treatment. Four full vital capacity breaths of pre-oxygenation before fast sequence induction may "de-nitrogenate" the terminal alveoli enough to maximise oxygenation. If four volume-controlled breaths are not achievable before inducing an obtunded patient, apneic oxygenation must be used [43].

Sedative hypnotics are widely used for inducing anaesthesia. In order to achieve a healthy equilibrium between the induction of anaesthesia and the resulting hemodynamic alterations, it is necessary to lower and titrate the standard induction dose. When compared to other tranquil hypnotics, ketamine [1 mg/kg] does not have the same effect on the body's systemic vascular resistance. Propofol is often used to induce anaesthesia, although it has the side effect of greatly lowering systemic vascular resistance. Patients with low blood pressure should get lower dosages of propofol [0.5-1 mg/kg] while sedated. In order to avoid vascular collapse, continuous volume resuscitation is required [44].

It takes around 45 seconds for a conventional fast sequence induction dosage of succinylcholine [1 mg/kg] to produce neuromuscular relaxation enough to permit endotracheal intubation. When succinylcholine isn't an option, rocuronium's non-depolarizing neuromuscular relaxant properties come in handy [e.g., burns, spinal cord injury, hyperkalemia]. Intubating circumstances comparable to succinylcholine may be achieved with a higher dosage of rocuronium [1-1.2 mg/kg] in around 60 seconds [45].

Aspiration may be prevented by immediately inserting an endotracheal tube into the trachea after induction. In order to successfully secure the airway of a trauma patient, rapid sequence induction [RSI] with direct laryngoscopy is used. While there is some debate over whether or not in-line stabilisation is effective for RSI, it is nevertheless wise to avoid unnecessary cervical spine movement as much as possible while doing a laryngoscopy. Spinal cord injuries after direct laryngoscopy seldom result in or exacerbate cervical spine injuries, which is good news [46].

The laryngoscopist has several options for enhancing airway patency. A low-cost and efficient airway adjunct, the gum elastic bougie may be useful in securing a difficult airway. One of the benefits of using video laryngoscopy during intubation is a clearer picture of the vocal cords. However, this does not ensure intubation success on the first try or decrease the amount of time needed to complete the procedure. When it comes to airway adjuncts, it's still best to have a small range of items with which you're already acquainted rather than a huge variety of unfamiliar items on hand. It is also crucial to have a backup plan, such as surgical airway management tools, on hand [See Clinical Practice Guideline for Trauma Airway Management] [47].

Communicating with the surgeon after endotracheal intubation of the trachea and verification of end-tidal carbon dioxide promotes a smooth and efficient surgical procedure. Potentially lowering the risk of aspiration is the placement of an orogastric tube at this time [48].

Upkeep of anaesthesia

Both inhalational volatile agents and complete intravenous anaesthetics may be used

for anaesthesia maintenance [TIVA]. In order to provide sufficient sedation/hypnosis and analgesia, both methods need to be properly titrated to the patient's hemodynamic profile. Assuring the administration of a sedative-hypnotic [e.g., propofol, benzodiazepine] and an analgesic [e.g., opioid] during TIVA may help reduce patient awareness and the acute pain response. Titrating the morphine dosage to the patient's hemodynamics is possible. Immediate measures must be taken to provide adequate intravenous access [e.g., large bore peripheral IV, intraosseous]. Extra intravenous access or an arterial line placement [if necessary for continuous monitoring of heart rate and blood pressure] may be done [49].

As a starting point for the rest of the resuscitation, sending a baseline set of labs, including coagulation tests and base excess, is recommended. Point-of-care testing [i.e., iSTAT results] should be validated with conventional laboratory tests. Monitoring the pattern of MAP is useful for directing anaesthetic maintenance and resuscitation. While there is some debate on what constitutes optimum blood pressure, research has linked [MAP] of less than 55 mm Hg to acute renal damage and myocardial injury after non-cardiac surgical anaesthetics. End organ perfusion is improved while uncontrolled bleeding is minimised by keeping MAP above 55 mmHg [50].

Isolated bouts of hypotension may significantly increase mortality in patients with traumatic brain injury. Systolic blood pressure should be maintained at >90 mmHg in individuals with proven or suspected traumatic brain damage [51].

Hemorrhaging ovarian cysts are possible. The hazards associated with hemorrhagic ovarian cyst [HOC] rupture include hemoperitoneum, decreased blood supply to essential organs, and sepsis because of the escape of blood and fluid into the surrounding belly and pelvis. Treatment to restore lost blood and laparoscopic surgery to stop the bleeding or remove the cyst may be required if the bleeding is severe [51].

Depending on the amount of blood loss, many surgical procedures may be performed on patients with ruptured HOCs. For the abdominal and pelvic regions, however, laparoscopy is the procedure of choice because of its accuracy. Fewer scars, less time, and less discomfort are other benefits compared to open surgery. [52].

The world over, a severe obstetric haemorrhage is still a primary cause of maternal death and morbidity. According to WHO research, between 25 and 30 percent of all maternal fatalities occur during or immediately after delivery. While antenatal, delivery room and postpartum care in industrialised countries have helped significantly reduce maternal mortality, rates remain stubbornly high in many developing nations. It is well-acknowledged that postpartum haemorrhage is a major cause of maternal mortality in low-income nations [53].

Massive obstetric haemorrhage has been the subject of much research into its aetiology, prevention, and treatment. As best we could, we've attempted to include the most recent findings and research in this compilation of the literature. While it is acknowledged that because of insufficient data, professional opinions will differ, the goal of this article is to present a realistic and pragmatic strategy for managing and the coagulopathy associated with it. There are a number of issues that make management difficult. The presence of amniotic fluid and the possibility of undetected bleeding both make it difficult to accurately assess blood loss. The extent of blood loss is obscured by pregnancy-related physiological changes [54].

Hemorrhaging may cause a variety of serious medical complications, such as ARDS, coagulopathy, shock, infertility, and necrosis of the pituitary gland [55]

Since obstetric patients with significant bleeding may quickly deteriorate, it is important to engage a senior anesthesiologist and the critical care team as soon as possible. A woman's life, not her uterus, should be prioritised during resuscitation [56].

In order to prevent hypothermia, warm fluids should be used to maintain resuscitation. If final therapy has already begun, then you may think about options like an arterial line, central line, or urine catheter. Resuscitation and fluid management must be started immediately after their insertion.

Indications for delivery, timeliness requirements, the degree of maternal hypovolemia, and obstetric history [such as a previous caesarean birth] all factor into the decision of which anaesthetic method to use [as in the case of APH].

Immediate resuscitation to restore tissue oxygen supply, as well as the detection, prevention, and treatment of hemostatic problems, are the primary goals of care.

One of the relative contraindications of regional anaesthetic is the existence of cardiovascular instability. If the sympathetic nervous system is blocked, hypotension from bleeding may become much more severe. Once hemostasis is confirmed and cardiovascular

stability is established, regional anaesthesia may be employed.

When an active epidural has been used during labour, this may be the best option. When it comes to controlling blood pressure [BP], a continuous epidural block is favoured over a spinal since it may be utilised for longer procedures. A thorough anaesthesia history, airway examination, antacid prophylaxis, and preoxygenation are all standard safety measures that should be done before any surgical procedure. In hemodynamically unstable patients, a fast sequence induction of general anaesthesia using cricoid pressure is the preferred method of anaesthesia [57].

Blood cells are saved during surgery

When it's believed that cell salvage CS may lessen the need for allogeneic [donor] red cell transfusions and/or severe postoperative anaemia, it's strongly advised that they be used. We advocate for the widespread availability of peri-operative cell salvage around the clock at all hospitals doing operations [apart from minor/day case ones] when blood loss is a known possible consequence [60].

Basics of intraoperative cell rescue

Gathering blood that has been lost after surgery is the first step in cell salvage. While being aspirated into a collecting reservoir through low-pressure suction, the blood is combined with an anticoagulant, often heparinized saline or acid-citrate dextrose, before being filtered. Centrifugation is used to separate the red blood cells from the total anticoagulated blood. After being removed from the patient, the red blood cells are cleaned with 0.9% intravenous saline and pumped into a bag. Multiple CS platforms may be found on the market. There is no significant difference between the methods in terms of the final result, which is the patient's red cells suspended in saline [61].

Heart, major vascular, major hepatobiliary, major spinal, major arthroplasty [especially revision hip replacement], major urological, thoracic, abdominal, and pelvic trauma, and major obstetric operations and haemorrhage are all examples of surgeries where cell salvage is routinely performed [62].

Although there are no hard and fast rules against CS, any possibility that the aspirated blood might be tainted by faeces, bacteria, or tumour cells should be considered a relative contraindication. The risks and potential rewards of CS should be weighed in these cases [63].

Large-scale transfusion of blood

There are several different definitions of MBT that have been published in the medical literature, including [64]:

Complete blood volume exchange in less than 24 hours

Whenever an ongoing need is anticipated, a transfusion of more than four units of packed red blood cells [PRBCs] within one hour is considered to be appropriate.

50% of TBV restored within 3 hours

Dangers associated with blood transfusions on a large scale

Rapid Volume Replacement-Related Issues

Failure to Restore Circulation: Lactic acidosis, systemic inflammatory response syndrome [SIRS], disseminated intravascular coagulation, and multiorgan failure are all consequences of inadequate blood flow.

Excessive use of resusitation

Overload of the circulatory system caused by a transfusion: The illness is well-known because it results from the fast transfusion of blood or blood products. Patients needing huge transfusions are at risk, although it is more prevalent in the elderly, young children, and those with reduced left ventricular function. Initial resuscitation of individuals with hemorrhagic shock involves the use of crystalloids and colloids. By transfusing patients with necessary components when blood and blood products become available, circulatory overload may occur [65].

Abdominal compartment syndrome may result from interstitial edoema caused by elevated hydrostatic pressure.

In anticipation of severe blood loss,

- Intravenous [IV] access with a large bore: Two peripheral intravenous [IV] cannulas [14/16 gauge] or a wide-bore cannula [insertion sheath] may be placed in the veins of the neck. Cannulation of the external jugular vein is a potential treatment option in critical cases.
- Surface and in-line fluid heaters are examples of warming equipment.
- The core temperature will be tracked continuously.
- Continual invasive monitoring of arterial pressure.
- Sufficient supplies of the necessary colloid [gelatins], crystalloid, infusion sets, and intravenous calcium preparations.
- Informing a blood bank of the impending need for their services due to substantial blood loss.

- Having enough staff to transport samples for analysis and collect blood and blood products is essential.
- Arterial blood gas [ABG] and thromboelastography are two examples of extremely desired point-of-care tests [TEG]. An hourly repeat of an ABG that includes Hb, electrolyte, and lactate values may help guide treatment.
- Pressure bags or rapid infusion pumps to expedite the delivery of fluids.
- Intensive care after surgery: Circulatory overload and hemodynamic/biochemical instability are common causes for the need for mechanical ventilation and regular monitoring of vital signs [66].
- Interventions for severe blood loss

A healthy individual will have the following vitals: a] Mean arterial pressure [MAP] about 60 mmHg, systolic arterial pressure 80-100 mmHg [in hypertension patients one may need to aim higher MAP]; b] Hb 7-9 g/dl c] INR 1.5; activated PTT 42 s d] Fibrinogen >1.5-2 g/L e] Platelets >50 109/ [67].

Conclusion

Since the process of managing perioperative bleeding is so dynamic, doctors need to be very cautious while dealing with it. Recognizing the most appropriate solution is crucial for treating this problem. It's possible that failing to do so might result in diminished efficacies of existing therapies or a worsening of symptoms. However, further study is needed to determine the true efficacies of each intervention, which will vary by the kind of operation. More studies should be conducted to examine the efficacies of the aforementioned methods outside of the fields of cardiac surgery and orthotopic liver transplants.

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